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Effects of Ultraviolet Radiation: Rays from the ultraviolet region of the spectrum may involve the transformation of radiant energy into chemical energy in the absorbing medium. When occurring, such a process is designed as a photochemical reaction.

Familiar examples of photochemical reactions are the photosynthesis of sugar by green plants, photography, and irradiation of milk to enhance the vitamin D content. Less well known are the enormous number of photochemical reactions, both organic and inorganic, which have been studied quantitatively in vitro and are well understood.

Basic to the knowledge of the action of ultraviolet light is the particular wavelength of the radiation in question because the wavelength characterizes the properties of the radiation -- i.e., its penetrability, its chemical effects, its energy content, and in the final analysis, its utility.

The energy transfer involved in photochemical reactions is best explained on the basis of the quantum theory. Certain sources of radiant energy emit photons. The photon is a discrete quantity or "quantum" of electromagnetic radiation and has associated with it an energy proportional to its frequency which is expressed as the product of a constant times its frequency. Hence, since the shorter the wavelength the higher the frequency, the energy of 1 photon at 3,000 Angstrom units, for example, will be twice as great as that of one at 6,000 A. This relationship explains why the very short roentgen ray is so much more destructive to matter than the ultraviolet ray.

In photochemical processes, (1) the radiation must be absorbed by the reacting substance, and (2) the absorption of radiation involves the capture of usually 1 photon (or more) per reacting atom or molecule. The mechanism of the action of ultraviolet radiation depends on the absorption of a quantum of energy by the substance with a shift in the valence electron, thereby activating the molecule for chemical reaction. With roentgen radiation the quantum absorbed is larger and other inner electrons become affected. Infra-red radiation, having small quanta of energy, increases the kinetic energy of the molecules and hence causes only a rise in temperature.

In this review, photochemical changes following ultraviolet radiation will be limited to substances of biologic and physiologic interest only. Of these, nucleoproteins and proteins are of paramount importance, since the former is one of the most essential constituents of the nuclear material, and the latter is abundantly present in the cytoplasm. Both absorb certain frequencies, leading to chemical reactions that produce disruption of the molecules and, which, if occurring in living tissue, cause injury or death of the cells.

Maximum absorption of ultraviolet radiations by proteins occurs at wavelengths of 2,800 A; the molecules undergo unfolding and breakdown. In vitro, albumin can be denatured, with a resultant coagulum of changed color and odor.

Other substances of protein nature respond similarly: fibrinogen is precipitated from solution; insulin loses its hypoglycemic effect; and enzymes are inactivated. In unicellular organisms, cilia, being protein in structure, cease motion with irradiation at 2,800 Å. In higher animals, only the epidermal cells are affected, since the penetrability of ultraviolet radiation is limited. It appears that the action on the protein molecule results from absorption by and disruption of the constituent cyclic amino acids, tryptophan, tyrosine, and phenylalanine. Radiations of frequencies below 2,300 Å are absorbed by all amino acids. The exact mechanisms here involved are still obscure, but probably deamination occurs.

To understand the reaction involving nucleoproteins, one must remember that these substances contain nucleic acid linked to certain proteins. The nucleic acid molecule is a complex one, built up of pentose, phosphoric acid, and the heterocyclic compounds, purine and pyrimidine bases. These two bases strongly absorb radiations in a narrow ultraviolet zone, the maximum being at 2,600 Å, with disruption of the rings. They alone account for the action of radiations from this region of the spectrum on nucleoproteins and, consequently on the cell nucleus.

Because of this property of nucleic acids to absorb specific radiations, ultraviolet rays have been employed as an investigative tool to disclose the secrets of the structure and metabolic events in a living cell. Briefly, brilliant results have been achieved, utilizing three main methods of approach: (a) Determination of absorption of radiations by a cell in a particular region of the spectrum and comparison with known absorption spectra. (b) Quantitative determination of known cell constituents in situ by determining the degree of absorption. This allows estimation of nucleic acid in the order of magnitude of 10^{-6} mg. (c) Comparison of absorption spectrum with an "action spectrum." The latter relates a series of wavelengths to their effectiveness in producing a given result.

With these methods in conjunction with the Kohler microscope, which utilizes an ultraviolet radiation source, nucleic acid components of the cell nucleus have been found to play a most important part in metabolic processes and in the complicated structure built up during cell division. The effectiveness of the microscope depends on the fact that since different constituents of a cell (protein or nucleoprotein) will selectively absorb given wavelengths, differences in structure can be visualized, without staining, by photographic means or by a fluoroscopic screen. Some of the important findings, presented fully in a monograph by Caspersson, are the following:

1. The nucleus has a high content of nucleic acid, up to 10 percent.
2. Nucleic acid becomes concentrated in the chromosomes, which are constructed of alternate segments of nucleic acid and protein.
3. Nucleic acid increases just before cell division and decreases at completion.
4. Ultraviolet irradiation is most destructive at the prophase, causing a break-up of the

chromatin material. 5. Mutations are most effectively produced by wavelengths of 2,600 A, again implicating nucleic acid in the process of cell reproduction. 6. Concentration of nucleic acid in the nucleus changes during functional activity of gland cells. In addition, the ultraviolet microscope affords a method of studying cell architecture in the living cancer cell.

The lethal effects of ultraviolet radiation on bacteria, fungi, viruses, and bacteriophage are well known. It has been shown that the action depends on the disruption of the constituent nucleic acid molecule by absorption of wavelengths in the region of 2,600 A. Mutations have been produced in Penicillium notatum to obtain richer yields of penicillin. The utility of ultraviolet radiation in air sterilization of schools, wards, and operating rooms is also well established.

Other biologic effects may find use in the field of immunology. In animals, irradiation appears to raise the antibody titer against typhoid and accelerate antibody formation when killed Eberthella typhosa and Escherichia coli organisms are injected.

What has been stated in relation to the biochemical processes concerned in the energy transfer of ultraviolet radiation in general can be applied concerning sunburn and suntan in which only epidermal cells are involved.

Erythema is produced by wavelengths below 3,200 A, the action spectrum displaying two maxima, 2,950 A and 2,500 A, which are thought to correspond to the absorption by protein and nucleic acid, respectively. The former zone is by far the most effective, and suggests a process of protein destruction.

The various events taking place in the epidermis after strong radiation (sunburn) can be followed histologically: 1. After a latent period of a few hours there is engorgement, enlargement of capillaries and intracellular edema. 2. Later, leukocytes leave the capillaries in the corium and migrate to the epidermis. 3. Twenty-four hours later degenerative changes in the stratum germinativum are observed. 4. After the active stage, proliferation of all epidermal layers takes place, including thickening of the corneum. 5. With fading of the erythema, pigmentation ensues.

This procession of events has been interpreted to be due to the release in the epidermis of substances with specific actions following cell injury by the photochemical disruption of the constituent protein. These are (1) a vasodilator H substance or substances, slowly released as a result of cell injury and producing a picture of inflammation rather than one of histamine effect; (2) a leukotaxin exerting a chemotactic effect on leukocytes and increasing capillary permeability; (3) a necrosin prolonging still further the destructive effect on the cells. Since fever may result after a sunburn, a pyrexin may also be assumed.

The second event following exposure to sun or other ultraviolet sources is tanning. Normally, melanin pigment can be found in the basal layers of the

epidermis. A few days after exposure, the basal cells are free of pigment, and melanin is found in the upper layers, including the stratum corneum, but the total epidermal pigment remains unchanged. This must mean a migration of old pigment toward the surface. Later, new melanin resulting from a specific photochemical reaction appears. In the first place, other means of cell destruction, such as friction, burn, and inflammation, can produce pigmentation; in the second place, the action spectrum for melanization is the same as that for production of erythema. Therefore, from the breakdown of cell protein it appears that there is produced some substance -- a melanotactic factor -- which is responsible for these changes.

On the other hand, it has been shown that a second mechanism for pigmentation also occurs. This is a true photochemical reaction and is known as a pigment-darkening reaction. Briefly, the existence of this second mechanism, distinct from the melanotactic process, rests on the following evidence; 1. The action spectrum ranges between 3,200 and 4,200 A, and pigmentation can be produced through glass. 2. There is no latent period; darkening occurs in a few minutes rather than in a few days. 3. A greater intensity of exposure is required. 4. Pigmentation occurs only in the presence of oxygen, in contrast to pigmentation following erythema. 5. Wavelengths above 3,200 A do not cause pigment migration. 6. Pigment darkening can be demonstrated on cadavers. These facts can be interpreted as a photochemical oxidation of bleached pigment (from previous tanning?) already in the skin. This explains why darkening is more in evidence with exposure to sunlight than to artificial sources of lower wavelengths. Incidentally, it has been observed that sex hormones also darken skin in previously tanned areas.

In summary, then, there are two mechanisms involved in suntan -- first, a release of a melanotactic factor causing migration of old pigment and formation of new pigment after a few days and, second, immediate photochemical oxidation of old bleached pigment in the epidermis by longer wavelengths.

Immunity to sunburn, as previously thought, is not due to melanization. Thickening of the stratum corneum effectively screens the erythema-producing radiations, and this is what mainly affords protection from further injury. This opinion is supported by several facts. The corneum is an excellent absorber of wavelengths below 3,000 A, and following an erythema, hyperplasia of the epidermis with thickening of the corneum has been demonstrated. In line with this, vitiligo patches and the skin of albinos are more resistant after exposure. The palm and soles, where the corneum is thickest, do not burn. Pigmentation, therefore, plays a distinctly minor role in protection against sunburn.

Sunburn of the eye, or photophthalmia, is due to an action similar to erythema of the skin, except that there is no pigmentation and the corneal cells do not proliferate to produce an immunity.

Minor physiologic effects of exposure to ultraviolet radiation, probably depending on the absorption of H substances, may be mentioned in passing --

for example, a transient and small decrease in blood pressure and a rise in gastric secretion. Red blood cells and platelets and blood coagulability have been reported to rise with repeated exposures, but these points are controversial. A negative nitrogen balance and increased excretion of uric acid have been reported in animal experiments, but other factors were not excluded.

Regarding the problem of abnormal sensitivity to ultraviolet radiations, the mechanism involving the release of H substances in the production of erythema appears to accord with accepted notions of allergy. This hypersensitivity is produced only by radiation frequencies below 3,200 Å. In other words, the action spectrums of normal erythema and hypersensitivity coincide; hence, it is considered reasonable to assume the same photochemical products in the former as in the latter. The marked reaction of the sensitive person may depend on either a greater sensitivity of his tissues or an allergen-antibody reaction which itself produces further cell damage with a pronounced erythematous reaction. The latter reasoning is supported by the fact that humeral transfer of photosensitivity is possible. When blood is taken from a susceptible person and injected intradermally into a normal one, the site of injection shows a marked reaction with exposure to rays under 3,200 Å. Such transfer may best be explained on the basis of circulating antibodies from the donor combining with antigenic protein breakdown products in the recipient. The combination of the two may give rise to an allergen-antibody reaction with the further liberation of H substances. Such a mechanism has not been actually proved as yet. In other conditions, hypersensitivity appears to depend in some fashion on local circulatory changes in the skin. Thus in hypertension, vasomotor instability, hyperthyroidism and in sympathectomized areas, erythema is readily produced by weak intensity of radiation. After removal of the thyroid gland, photosensitivity disappears.

Recently, a report has been published on the efficacy of pyribenzamine in reducing photosensitivity to erythema-producing rays. This evidence is another link in the concept implicating the action of H substances in ultraviolet radiation. It is of great theoretical and practical interest to find whether the antihistamine drug can prevent sunburn in normal people.

The presence of certain photodynamic dyes has long been known to exaggerate the effect of these rays in vivo. In vitro, certain salts of heavy metals and certain dyes like eosin can be shown to potentiate the effect of nonlethal intensities of radiation on unicellular organisms, with resulting death. Apparently, the dye molecule becomes activated by the absorption of 1 photon and is then capable of transferring the captured energy to a suitable substrate, resulting in a chemical reaction. When dyes are injected into animals, severe erythema and death can be readily produced by exposure to ultraviolet rays. Drugs of the sulfonamide class fall into this category, and one must be aware of this fact in therapy. Porphyrin has also been incriminated as a photodynamic dye, since clinically, porphyrinuria does occur. In vitro, this substance acts as a light sensitizer. However, all good evidence argues against its clinical importance: 1. In animals enormous amounts, far exceeding

clinical levels, need to be injected to produce a photodynamic action. 2. Porphyrin requires the presence of oxygen, whereas the erythema of light sensitization proceeds without it. 3. The action spectrum for porphyrin differs from that of erythema.

An intensively studied and well known photobiochemical reaction is that relating sterol metabolism to the vitamin group. The plant sterol ergosterol, when irradiated with ultraviolet rays, absorbs wavelengths under 3,200 A and undergoes a complicated reaction, yielding, among other substances, an active compound, vitamin D². The animal sterol 7-dehydrocholesterol undergoes a similar change, yielding vitamin D³. The latter is more potent against rickets than the former. Evidently, the provitamin is present in the corneum and upper layers of the epidermis, since radiations below 3,200 A are effectively screened. Other D vitamins can be produced by starting with different precursors, but they all are similar in respect to the breaking of the bond between the carbon atoms in positions 9 and 10.

The effect of ultraviolet radiation on rickets through the mediation of a chemical substance produced locally and exerting a distant action suggests that possibly a similar mechanism may account for the favorable influence of ultraviolet rays in other systemic conditions. Extrapulmonary tuberculosis may be one such condition.

Deleterious effects of ultraviolet radiation have recently been suspected as leading to skin cancer. The evidence is mainly statistical but impressive: 1. The incidence of skin cancer is greater in lower latitudes. 2. Of skin cancers, 95 percent occur on exposed regions like the face and hands. 3. There is a greater incidence in outdoor than in indoor workers. 4. White persons are more affected than Negroes. Moreover, neoplasms have been induced in mice by radiations below 3,200 A, although it must be pointed out that sarcoma, rather than carcinoma, has resulted. Moreover, corneal tumors, which occur mainly in the southern population, can also be produced in mice. The mechanism is unknown. Possibly one or all of three methods may be responsible: 1. Mutation of cell chromosomes by action of the rays on nucleic acid. 2. Carcinogenic breakdown products of cell substances. 3. Presence in the skin already of precancerous lesions. (Arch. Phys. Med., Jan. '48 - E. E. Gordon)

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The Initiation of Cellular Infection by Influenza and Related Viruses: In a study to determine the nature and significance of the phenomenon of red-cell agglutination by influenza and related viruses, several aspects of the work have been brought together recently in a provisionally satisfactory fashion to form a solid stepping-off point for further work. It has been found that the influenza viruses contain as an integral part of their surface structure an enzyme which is adsorbed by and eventually destroys certain specific molecular groupings of a mucin which forms part of the cell surface. Unless this process can occur, infection of the cell cannot take place. The authors have obtained and partially

purified a soluble enzyme which, by destroying the specific adsorptive power of the mucin in the cell surface, prevents adsorption of the virus, and infection.

These findings appear to open up a number of possibilities of high potential importance, notably a logical approach to virus chemotherapy.

Virus Hemagglutination. Very soon after it had been found that influenza viruses could multiply in the allantoic sac of chick embryos and produce fluids containing large amounts of virus, Hirst, in 1941, observed that such fluids agglutinated red cells. It was immaterial whether chicken, human, or guinea-pig cells were used. With a typical active allantoic fluid any of these cells would be agglutinated up to a dilution of a few hundreds.

Detailed investigation of the phenomenon soon made it clear that the agglutination was a function of the virus particles themselves. If an active virus preparation is treated with an excess of red cells in the cold, almost all its infective power is carried down with the cells on centrifugation. Agglutination is essentially similar to agglutination by an immune serum. Instead of the bridges made by union of single antibody molecules with antigen on two different cells, there are virus particles functioning in similar fashion binding the red cells into rapidly settling aggregates. With suitable quantitative methods it is easy to adapt hemagglutination to the titration of virus and antibody, and most of the detailed present-day work on the epidemiology of influenza, on the preparation of vaccines, and on tests of their effectiveness makes use of such methods.

The first evidence that viruses other than those of the influenza group would agglutinate red cells was obtained with Newcastle disease of fowls and shortly afterwards with fowl plague. Then in 1945 Levens and Enders found that mumps virus was also active. All these viruses can be shown to cause agglutination as the agglutinating agent and the same type of receptor on the red cells is involved. The finding from the laboratory of the author and co-workers that vaccinia and mouse-pox (ectromelia) viruses also agglutinate certain red cells is not directly related to the question; in these reactions a soluble substance, not the virus, is concerned.

The first indications of the real nature of the hemagglutination reaction with influenza virus were obtained by Hirst in two fundamental investigations. In the first, in 1942, he showed that red cells to which virus was adsorbed in the cold could be freed of the virus by holding for a few hours at 37° C. With the elution of the virus the cells became stable in suspension and were not agglutinated by fresh virus. The eluted virus retained its full infective and hemagglutinating power and could be used successively to treat repeated quantities of red cells. Hirst recognized that this capacity of the virus to destroy a virtually unlimited amount of red-cell "receptor" was characteristic of an enzyme reaction.

In the second, in 1943, Hirst showed that essentially similar reactions took place in the excised ferret lung. In a suitable set-up, virus run in through the trachea was rapidly adsorbed and held by the lung for from 2 to 5 hours before elution occurred with almost complete reliberation of the virus. Hirst deduced from this phenomenon that the susceptible cells of the respiratory tract reacted similarly to red cells, and that the essential first stage in the process of infection was adsorption to and enzymic destruction of some specific component of the cell surface. The fundamental problem then was to identify this vital component of the cell surface.

The author summarizes the results of this investigation as follows:

The hemagglutinating action of the mumps-influenza group of viruses has been shown to be due to the presence of an enzyme as an integral part of the virus particle surface.

The substrate of the enzyme is a mucin present in the surface layer both of erythrocytes and of the cells susceptible to infection by influenza viruses.

By the use of an enzyme derived from the causative organism of cholera and active against the same substrate, cells can be rendered insusceptible to infection by viruses of the group. (Lancet, 3 Jan '48 - F. M. Burnet)

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Chemotherapy and Pharmacology of Aerosporin: Aerosporin (News Letter of 21 November 1947) is an antibiotic produced by an aerobic spore-bearing bacillus identified as Bacillus aerosporus. In 1947 Ainsworth et al. described the isolation of this organism from soil and air and noted its probable identity with Bacillus polymyxa. They devised a method for producing the antibiotic in half-strength broth containing sucrose or glucose with traces of manganese, and isolating it by adsorption to a suitable charcoal and by subsequent elution with aqueous acetone after treatment with sulfuric acid. Being basic, the antibiotic is first eluted as the sulfate; it is further purified by conversion to the helianthate and then to the hydrochloride, which is amorphous and hygroscopic.

Throughout this report the concentrations are stated in terms of aerosporin standard 1947, containing 10,000 "units" per mg. A convenient method of assay has been by a dilution technic using Escherichia coli as the test organism. In one modification of the test the dilutions are read after about three hours' incubation at 37° C. Material containing 10,000 units per mg. may, for practical purposes, be considered to be the pure hydrochloride; but final claims of this kind are best withheld until the chemical and physical properties of the substance, and if possible the constitution of the molecule, are known. The preliminary observations of Tudor S. G. Jones, not yet published, have shown that leucine, threonine, and a third, unidentified basic amino acid enter into the constitution of aerosporin.

This report is mainly concerned with the effectiveness of aerosporin in experimental infections with Gram-negative bacteria, against which it has a highly selective action, and particular attention has been given to the infections in which established chemotherapy has hitherto been largely unsuccessful, perhaps the most important being those causing whooping cough and typhoid fever. The evidence presented takes the form of in-vitro and in-vivo comparisons with streptomycin and occasionally other chemotherapeutic agents.

In the test-tube, aerosporin is from ten to many hundred times more active than is streptomycin against several Gram-negative pathogens. It has no action, however, against Mycobacterium tuberculosis.

Impressive features of aerosporin which emerge from this study are its bactericidal power, reflected in the protection afforded to infected animals, and its reluctance to produce resistant strains. To mice infected experimentally with Hemophilus pertussis aerosporin gave more protection than streptomycin when the two antibiotics were administered in doses proportional to their in-vitro titres. The bactericidal nature of aerosporin is illustrated in the high protection afforded by only two doses, each of 100 micrograms compared with the poor protection resulting from 1250 micrograms of streptomycin given twice daily for five days. Again, the rapid elimination of the infection by aerosporin contrasts with the slighter protective action of the sulfonamides. Long treatment with large doses of sulfadiazine or sulfathiazole gives surprisingly good results but does not eradicate the infection, since deaths occur after treatment has stopped. Shorter treatment with sulfonamides when compared with aerosporin therapy emphasizes the superiority of aerosporin. The chemotherapeutic activity of aerosporin against H. pertussis suggests that it may prove of value in whooping cough, and the clinical trial in 10 cases reported by Swift in 1948 supports this view. (See following article.)

Eberthella typhosa is more sensitive in vitro to aerosporin than to streptomycin. In mice with an overwhelming infection, which led to the death of untreated controls within six hours and in which continued penicillin and sulfadiazine therapy was without effect, aerosporin produced higher survival rates than streptomycin. Aerosporin therefore may be expected to exert a demonstrable effect in typhoid fever.

The high activity of aerosporin in the test tube against strains of Hemophilus influenzae, and its equal efficiency with streptomycin in the treatment of intracerebral infections of mice, indicates that aerosporin may be at least as effective as streptomycin in H. influenzae meningitis of man. Intrathecal injection in rabbits shows that aerosporin is well tolerated and that a therapeutic concentration in the cerebrospinal fluid may be maintained for 24 hours after a single dose.

Other infections of which the causal organisms are highly aerosporin-sensitive and that may therefore be expected to respond to aerosporin are

those due to Escherichia coli and organisms of the genera, Pasteurella, Salmonella, and Shigella. Vibrio comma, Brucella abortus, and Pseudomonas aeruginosa (Bacillus pyocyaneus) infections may also respond to aerosporin. Though treatment of experimental Ps. aeruginosa infections was unsuccessful, this organism is sensitive in vitro to concentrations of aerosporin that can easily be maintained in man.

The fact that aerosporin exerts its bactericidal action at the same speed in broth and in water, together with the absence of observed morphological changes in bacteria submitted to sublethal doses, suggests that it acts by interfering with a system more immediately vital than reproduction. The bactericidal action of aerosporin takes place more slowly in serum than in broth and is influenced by the number of organisms present; these facts suggest that successful therapy may depend on the maintenance of continuous adequate serum levels. Given parenterally it disappears from the blood-stream quickly. Hence, for man the suitable spacing of doses seems to be from 3 to 4 hours.

Aerosporin does not enter the blood stream from the alimentary canal, and therefore it is useless to give it by mouth in systemic infections. Combined oral and parenteral administration may, however, prove most effective in intestinal infections, for aerosporin given by mouth eliminates the sensitive organisms from the gut.

Disadvantages of aerosporin therapy may be seen in the observations that in the normal animal the antibiotic does not appear in the cerebrospinal fluid nor is it excreted either in bile or in urine, at least not in a biologically active form. The available evidence indicates that the aerosporin molecule is large and does not pass the kidney barrier.

The difficulty with which a strain of E. typhosa could be induced to develop resistance to aerosporin is a most significant feature. After 28 passages in a fluid medium there was no alteration in the sensitivity of the strain, and 34 passages on solid media were necessary to increase its resistance sixfold. Under identical conditions only 10 passages were necessary on solid streptomycin media to increase its resistance about two hundred times.

The acute intravenous toxicity of aerosporin of near chemical purity is 6.14 mg. per kg. - i.e., about thirty times as great as that of streptomycin base. However, in experimental infections with H. pertussis and E. typhosa, aerosporin is so much more effective than streptomycin that the therapeutic index of aerosporin is actually higher. A more realistic therapeutic index can be obtained by relating the blood-aerosporin level corresponding to the minimal lethal dose, LD₅₀, and the average therapeutically effective blood level. Since the former averages from 30 to 40 micrograms per ml. and the latter from 0.2 to 0.4 micrograms per ml., there is an ample margin of safety. Aerosporin is non-hemolytic, and it has about the same toxicity to leucocytes as penicillin.

Aerosporin concentrates are more or less contaminated with an anti-diuretic principle and a substance which damages the renal tubules. Purified material is free from the second factor, and the first has not been shown to act in man. (Lancet, 24 Jan '48 - G. Brownlee and S. R. M. Bushby)

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Treatment of Whooping Cough with Aerosporin: The prompt and decisive action of aerosporin in experimental Hemophilus pertussis infections raised the hope that it might prove equally successful in the treatment of whooping cough, and a clinical trial was initiated.

Aerosporin was used in the treatment of a series of 10 unselected consecutive cases of whooping cough in children, aged from 1 month to 2 and 1/2 years, Early and late, mild and most severe, and 2 moribund cases were included.

From the work of Brownlee and Bushby it appeared that blood levels of from 0.1 to 0.2 microgram per ml. were likely to prove chemotherapeutic and that a dose of from 0.2 to 0.4 mg. per kg., in terms of aerosporin standard 1947, would maintain this level in man for four hours. Only batches containing minimal quantities of the albuminuria-producing factor were used; one batch (that used in the last case) was free from this impurity. For these reasons the first 3 patients received only 0.2 mg. per kg. four-hourly; but, since no toxic signs developed, the dosage was raised to 0.4 mg. per kg. four-hourly for mild and moderately severe cases. The observation that a very severe case did not respond to 0.5 mg. per kg. four-hourly but did respond to 0.8 mg. per kg. three-hourly led to the adoption of 0.8 mg. per kg. four-hourly as the routine dosage. In 3 cases a three-hourly dose schedule was adopted as an alternative to an increase in dose. The course covered five days.

Since Brownlee and Bushby have shown that a single dose of aerosporin protects 90 percent of animals experimentally infected with many lethal doses of H. pertussis, the dosage used in this series may be unnecessarily large. This is a subject for further inquiry.

The aerosporin was supplied in sterile sealed ampoules as a freeze-dried colorless hydrochloride containing the equivalent of 4 mg. of aerosporin standard 1947. The batches varied in purity from 70 percent to 80 percent and had been tested for sterility and for freedom from pressor bases and histamine-like impurities previously.

The progress of the disease was judged by observing prominent clinical features, such as the occurrence and intensity of postparoxysmal apnea, vomiting, whooping, and temporary or permanent cyanosis. Frequent differential white-cell counts and estimations of erythrocyte-sedimentation rate, and weekly radiography of the chest were used as a guide to progress and to detect complications. In view of the possibility of renal tubular damage, due to the presence of an impurity, repeated observations of renal function were necessary. A

record was made of fluid intake, urinary output, and daily weight, and the urine was examined chemically every day and microscopically less often. Blood urea was estimated at the start of therapy and later if albuminuria appeared. Though H. pertussis was isolated in only 1 case, a lymphocytosis was observed in all cases.

In most cases a therapeutic effect was observed within the first 48 hours, suggesting that the toxins of the causal organism were suppressed at the source. Vomiting, periods of apnea, cyanosis, and the frequency of paroxysms were first modified; and, as might be expected from observation of the normal course of pertussis, whooping and cough were last to disappear. Patients in which treatment was begun within a week from the onset of whooping enjoyed an uncomplicated recovery irrespective of the severity of the disease or the age of the patient. When treatment was not begun until a week after the onset of whooping the effects appeared less favorable. A probable explanation for the difference lies in the presence or absence of secondary invaders in lung parenchyma. Not only is a greater extent of lung damage to be expected as a result of the secondary invasion, but also the resulting lesions must be expected to favor the establishment and survival of pathogenic bacteria.

The expected damage to renal tubules, first observed in the animal experiments and known to be due to a contaminating substance, took the form of a transient albuminuria. Observed in 9 of 10 patients it varied from a trace to 0.4 Gm. of albumin per 100 ml. In 7 of these it disappeared within a week; in another it persisted for ten days and was associated with an Escherichia coli pyelitis. In one instance in which the child received two courses, the albuminuria disappeared within a week, reappeared with the second course, and again disappeared within a week. Finally, in the last case, albuminuria only appeared after the substitution, on the last day of the course, of an impure batch for a batch known to be free from the factor.

Gross hematuria was not observed; but, microscopically, red blood cells were found in 4 cases in which granular and hyaline casts were also present. Pus cells were encountered in 3 female patients, in 2 of whom E. coli was isolated from the urine on culture.

Sugar and acetone appeared in the urine in 3 instances, together with the albuminuria, and persisted in 2 cases for a day and in 1 case for two days.

A temporary rise of temperature to from 99 to 100° F., starting on the second day of aerosporin therapy and lasting two days, or as long as aerosporin was given, developed in 7 cases. In the last case it was not observed on the four days during which pure material free from renal-tubule-damaging factor was given, but appeared after the impure material was given for a day. No other toxic sign was seen.

As happened in this series, the optimal therapeutic effect of aerosporin may be expected to be exerted when treatment is started before secondary

infection is present. Benefit can, however, be expected after secondary invasion, as occurred in one case. The results show that aerosporin alone is effective in whooping-cough, especially in early cases. It is nevertheless not reasonable to assume that aerosporin alone is the treatment of choice in late cases. In such instances a combination of aerosporin and a sulfonamide may be more effective. (Lancet, 24 Jan '48 - P. N. Swift)

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Cancer of the Stomach in Patients with Pernicious Anemia: Among some clinicians there is a belief that patients suffering from pernicious anemia are more likely to develop gastric carcinoma than normal individuals in the same age groups. Kaplan and Rigler have reported 18 cases of cancer and 17 simple tumors in 259 cases of pernicious anemia.

During the past two years 15 of the author's patients with pernicious anemia have been observed by radiograph and gastroscopy in the hope of detecting carcinoma of the stomach, if present, at an early stage. The cases were not selected, nor did they form a series. These observations have resulted in the discovery of three cases of carcinoma in patients free from symptoms and clinical signs: one was certainly operable and one probably operable when first detected. One further patient has been found to have a leiomyoma. Two other patients are under particular observation because of a persistently high blood sedimentation rate; one of these has a nodule of uncertain nature in the pyloric antrum.

The total patients observed were made up of 11 females and 4 males. Radiological examination consisted of a barium swallow, fluoroscopy, and, usually, a film. Gastroscopy was performed with the Hermon Taylor or Schindler instrument if the radiological report was doubtful, if there was failure to improve clinically, and once when there was a persistently high sedimentation rate in the absence of abnormal radiological findings. It must be emphasized that no patient had dyspepsia when examined.

The patients developing carcinoma were all over the age of 65, and had been treated for pernicious anemia for ten years or more. With increasing age abnormal stomachs would be expected to become more susceptible to malignant change; and liver treatment, by keeping patients alive, may predispose to this form of cancer.

Periodical x-ray and gastroscopic studies are strongly indicated in all cases of Addison's anemia in remission or relapse. (Brit. M. J., 17 Jan '48 - W. A. Bourne)

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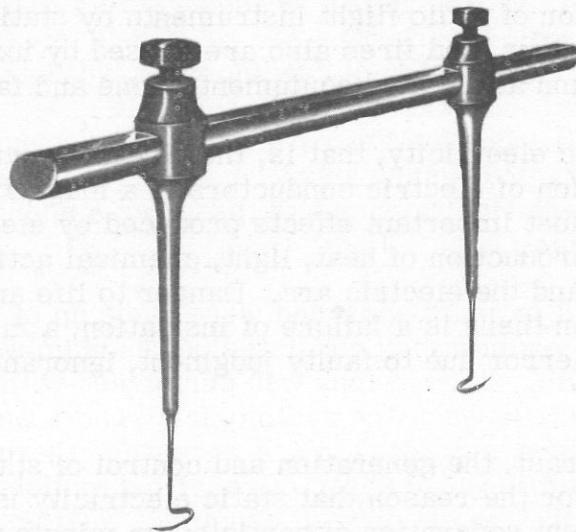
A New Device for Easier Skin Suturing: Frequently, when making repairs of the borders of skin wounds, surgeons apply hemostatic forceps at both ends,

distribute others along the wound, handle the borders with toothed forceps and feel surprised upon obtaining a defective scar.

The author has stressed the importance of eliminating the use of forceps in handling skin borders, because they destroy cellular groups which have to be repaired with fibrous tissue which, added to that generated to repair the original wound, will produce an unsatisfactory result.

He has also pointed out the necessity of making the borders coincide exactly with one another, in order to facilitate the suture technic. With this in mind, and following the principle that the application of two hooks, one at each end of the wound, exerting an outward pull can effect this, the author has constructed a device, the Dermo-Coaptor. This instrument (1) holds itself in place, (2) exerts the desired outward tension, (3) coapts the borders satisfactorily, and (4) eliminates the need of an assistant. It is illustrated below.

The device consists of a rod on which slide two arms, each having a hook perpendicular to its axis. The arms are held in place, at will, by means of knurled-head set screws which are tightened against the flat section of the rod. The device is made of a nickel-steel alloy which makes it rust-proof. Each instrument is constructed with two rods, a short one for plastic work, and a long one for general surgery.



To apply it, one of the hooks is placed at one extreme of the wound, and the other tightened at the point which produces the desired tension and the correct coaptation of the skin borders.

With its use, the author has simplified the technic and turned suturing into an exact and less time-consuming procedure. (Plastic and Reconstructive Surg., Jan. '48 - M. Gonzalez Ulloa)

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Electrical Hazards: Observers instinctively question why some victims who suffer great physical damage from contact with high-voltage circuits survive, whereas others are killed instantly by seemingly casual contact with a low-voltage circuit.

Explosions and fires caused by electricity, and electrocutions commonly are classified as originating from power circuits or from static electricity. For safety purposes, static electricity and dynamic electricity can be differentiated only by the mode of generation. Death or damage to property can occur only when electric charges move. Transfer of electric charges, regardless of their origin, constitutes an electric current, and it is the flow of current that gives rise to damage.

Costly explosions and fires caused by static electricity occur frequently in grain elevators, oil refineries, munitions plants, paint factories, motion-picture projection booths, hospital operating rooms, textile mills, printing establishments, gas-filled basements, and when petroleum products are discharged from tank cars or trucks. Many aircraft disasters, such as crash landings, fires, and explosions in the air, are attributed to static electricity. The United States Armed Forces report that some 25 percent of airplane casualties in the Army and Navy during the recent war, at home and abroad, resulted directly from obliteration of radio communications and from the erratic behavior of radio flight instruments by static interference. Electrocutions, explosions, and fires also are caused by insulation failures in power machinery and associated equipment, home and farm appliances, and the like.

Dynamic electricity, that is, the electric current of industry, is generated by the rotation of electric conductors in a magnetic field or by chemical action. The most important effects produced by electric power currents are the continuous production of heat, light, chemical action, magnetic forces, mechanical forces, and the electric arc. Danger to life and damage to property eventuate only when there is a failure of insulation, a misapplication of electric equipment, or an error due to faulty judgment, ignorance, lapse of memory, or carelessness.

By contrast, the generation and control of static electricity is not well understood for the reason that static electricity is generated by the contact and subsequent separation of particles or minute projections of insulating materials, or by the tearing apart of water droplets. Everyone is familiar with the accumulation, noticeable especially in cold dry weather, of electric charges as he scuffs a carpet, and with the consequent shock and spark when his hand approaches a grounded metal object. Usually the spark is harmless, and the shock is annoying only because it is unexpected. But in an industrial plant such sparks may be produced in a space containing combustible gases or dust, and there they constitute a very serious explosion hazard. The voltages which result from rapidly moving belts, conveyers, paper stock, fabrics, and rubber-tired vehicles, as well as those which originate at nozzles from rapidly issuing steam and gases, attain amazingly high values under favorable conditions. Electric charges also may be produced when petroleum products flow through pipes and hoses under conditions of high velocity and violent turbulence. The accumulation of these charges at terminal tanks under favorable conditions may produce voltages of from a few thousand to as many as 75,000 volts. Surprising as it may seem, a person can electrify his body to as high as 10,000 volts merely by scuffing over a woolen rug, and consequent discharge may

cause a spark of sufficient intensity to ignite a gas jet or discharge an electrical blasting cap.

Lightning, the most terrifying of frequent natural phenomena, is the result of vast accumulations of electric charges and their sudden discharge. The static electricity generated in a thunderhead is believed to result from the sudden tearing apart of water droplets as they are caught in the violent turbulent updrafts which are always present in thunderheads. Static electricity is important mainly because of its capricious behavior, because it may be a source of annoyance, and because it may produce fire or explosion.

Although the energy in the lightning discharge is small, the rate of energy dissipation is tremendous. The resulting pulse of current and the accompanying mechanical forces may produce almost unbelievable damage in the immediate vicinity of the stroke. Just prior to a lightning stroke the electric field is intense, and very large numbers of electric charges accumulate on near-by objects. At the instant the stroke occurs, the electric field between the cloud and the earth collapses, thereby releasing these bound charges, which, in turn, may produce secondary discharges. The secondary discharges are called induced strokes and, though of much less intensity than the main stroke, may break down commercial electrical insulations, or, if they result in open sparks, cause fire or explosion. Protection against lightning is afforded by lightning rods and gaps, ground wires, bonding wires, and lightning arresters. The discharge of static electricity is similar to lightning flash, except that the charges and voltages are very much smaller. Methods for the control of static electricity in industrial installations depend upon neutralizing or discharging the static electricity, so that no spark is produced.

Despite years of painstaking research and scientific development which have increased greatly the safety of electric machinery, equipment, and home appliances, and despite inspection of new installations which has done much to guard against misapplications, poor workmanship, or defective materials, shock and fire hazards still may develop from a variety of causes.

After all technical precautions have been taken, the second line of defense is education. The public must be warned continually against using defective or wornout machines, equipment, and appliances. The slightest shock must be regarded as having potentialities of death; the device must be disconnected immediately and inspected competently. The public must be educated to employ extreme caution when using electric machines and appliances in hazardous locations. For example, moisture and adjacent grounded objects create serious hazards and those responsible for safety must inculcate the habit of standing on some insulating material, such as a rubber mat, a dry board, or a pile of canvas, when electric equipment is being handled in wet basements or inside metal tanks. It must become axiomatic that an electric fixture or appliance never should be touched by anyone in a bathtub.

The frequency of electrical fatalities in the United States in recent years is fairly constant at 1/100,000 of the population. This figure is only a rough estimate, because it is difficult to obtain accurate information on a nationwide basis.

In September 1946, the Committee on Article 250, National Electrical Code, prepared a comprehensive report on electrical accidents, with special reference to reducing electrical hazards by proper grounding. The table below, a compilation of low-voltage accidents taken from this report, is based upon newspaper clippings for the years 1936, 1937, and from 1940 through 1942. Electrical accidents are classified according to the voltage of the circuit involved. Accidents on circuits below 600 volts are called low-voltage accidents.

	Others																								Totals							
	Home and Farm												Under 150					Over 150														
	1936		1937		1940		1941		1942		1936		1937		1940		1941		1942		1936		1937			1940		1941		1942		
	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF	F		
Bathtub:																																
Contacting cords.....	0..	2..	0..	2..	0..	4..	0..	2..	1..	6..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	16		
Contacting portable outside.....	0..	5..	0..	6..	1..	3..	0..	5..	0..	6..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	25		
Portable immersed.....	0..	2..	0..	5..	1..	6..	0..	2..	0..	5..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	20		
Extension cord:																																
Socket or plug.....	1..	1..	0..	8..	1..	9..	2..	9..	0..	7..	0..	4..	1..	5..	0..	4..	0..	2..	1..	7..	0..	0..	0..	0..	1..	0..	0..	0..	6..	57		
Cord.....	0..	4..	3..	6..	2..	5..	1..	10..	3..	10..	2..	4..	2..	5..	0..	6..	3..	3..	1..	4..	0..	0..	0..	0..	0..	1..	1..	4..	18..	62		
Floor and table lamp:																																
Frame.....	1..	3..	0..	3..	0..	5..	0..	9..	1..	4..	0..	0..	1..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	3..	25		
Conductor.....	1..	0..	3..	2..	0..	4..	0..	1..	1..	3..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	6..	10		
Vacuum cleaner:																																
Frame.....	0..	0..	0..	0..	0..	0..	0..	2..	0..	2..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	5		
Conductor.....	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	0		
Flatiron; toasters:																																
Frame.....	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1		
Conductor.....	0..	0..	1..	0..	0..	1..	1..	1..	0..	1..	0..	0..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	3..	3		
Washing machines:																																
Frame.....	0..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1		
Conductor.....	0..	0..	0..	1..	0..	1..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	3		
Refrigerators:																																
Frame.....	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	0		
Conductor.....	0..	0..	0..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0		
Portables (miscellaneous):																																
Frame.....	1..	0..	0..	1..	0..	0..	0..	2..	2..	2..	0..	1..	0..	2..	0..	2..	0..	3..	0..	1..	0..	2..	0..	1..	1..	0..	2..	6..	0..	6..	23*	
Conductor.....	2..	0..	0..	0..	0..	2..	0..	2..	0..	3..	0..	0..	0..	0..	0..	1..	0..	0..	1..	0..	0..	0..	0..	0..	0..	1..	0..	0..	3..	9		
Drills, Sanders, saws, and so forth:																																
Frame.....	0..	2..	0..	0..	0..	1..	1..	2..	0..	2..	0..	0..	1..	5..	6..	7..	2..	16..	2..	8..	1..	3..	0..	0..	1..	0..	1..	0..	13..	48**		
Conductor.....	0..	0..	0..	0..	0..	1..	1..	1..	0..	0..	0..	0..	0..	1..	0..	2..	0..	1..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	1..	6	
Touching live current-carrying parts.....	2..	1..	7..	5..	4..	6..	4..	5..	9..	4..	8..	6..	10..	1..	11..	3..	11..	3..	17..	2..	2..	1..	15..	3..	11..	1..	35..	0..	19..	46..	165	
Shocks from permanently wired equipment.....	0..	1..	1..	1..	0..	0..	1..	4..	0..	1..	2..	2..	0..	1..	0..	1..	0..	3..	0..	1..	0..	0..	0..	0..	0..	2..	2..	0..	6..	19		
Energized nonelectric equipment.....	4..	0..	3..	6..	1..	1..	2..	2..	3..	5..	0..	2..	1..	1..	1..	1..	2..	3..	0..	1..	0..	0..	1..	0..	1..	4..	1..	1..	22..	26		
Repairing testing electric equipment.....	0..	2..	2..	6..	1..	2..	0..	8..	1..	2..	1..	0..	0..	0..	0..	5..	2..	2..	0..	2..	0..	1..	2..	2..	7..	0..	6..	1..	12..	10..	57	
Doing a wiring job.....	0..	0..	0..	5..	0..	1..	2..	0..	1..	3..	4..	0..	1..	1..	0..	2..	1..	5..	0..	4..	1..	3..	0..	4..	0..	1..	8..	0..	1..	9..	37	
Welding machines.....	0..	0..	0..	0..	0..	0..	0..	1..	0..	0..	0..	1..	0..	5..	0..	9..	2..	12..	0..	9..	0..	0..	0..	0..	0..	0..	0..	0..	0..	2..	37	
Crane, trolleys, and the like.....	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	6..	26	
Grasping wires outside.....	6..	1..	5..	0..	5..	3..	8..	3..	3..	1..	0..	3..	3..	1..	0..	2..	0..	1..	1..	1..	0..	0..	0..	0..	1..	0..	2..	0..	1..	32..	20	
Working in mine.....	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	4..	85	
Electric fence.....	3..	1..	1..	3..	2..	3..	0..	3..	1..	7..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	0..	7..	17	
Shocks, no details or not clear.....	0..	0..	0..	0..	0..	2..	0..	2..	1..	2..	3..	1..	2..	3..	1..	6..	1..	1..	1..	7..	0..	1..	1..	3..	0..	7..	1..	8..	11..	46		
Total.....	21..	25..	27..	60..	19..	61..	23..	76..	24..	82..	16..	26..	18..	41..	10..	59..	17..	65..	10..	62..	5..	17..	6..	38..	7..	47..	14..	113..	5..	77..	220..	849

NF—Nonfatal.

F—Fatal.

* Includes five cases required to be grounded by present code rules.

** Includes 31 cases required to be grounded by present code rules, two cases where grounding means were provided and not used, and four cases where intended grounding means was the direct cause of the shock.

These are subdivided further into those under 150 volts and those over 150 volts but less than 600 volts. Especially to be noted is the substantial number of accidents which occurred in the home or on the farm, many of which involved bathtubs, extension cords, and lamps. Accidents involving portable tools are of particular interest because of the recent change in grounding recommendations. A similar tabulation, also based on newspaper clippings, of high-voltage (circuits of more than 600 volts) accidents, excluding utility cases, for the years from 1940 to 1942 inclusive, is given in the adjoining table. How many of the actual total of electric shock cases have been reported in the newspapers, it is difficult to say. A check made in one state indicated that, so far as fatal shock cases were concerned, the proportion reported in the newspapers was high. The small number of nonfatal shock accidents indicates that relatively few nonfatal electric shock accidents are reported in the newspapers. Electrical accidents from 1942 through June 1946, reported by the California Industrial Accident Commission, amounted to 165 fatal and 2,030 nonfatal accidents. Of these, there were 56 fatal and 1,745 nonfatal accidents occurring from low voltages; 42 fatal and 158 nonfatal accidents occurring from contact with high voltage lines (crane booms, mobile equipment, steel cables, and the like). It is mandatory for all industries in California to report electrical accidents, both fatal and nonfatal. These figures are of particular interest because they show the proportion between the fatal and the nonfatal shocks.

	1940		1941		1942		Totals	
	NF	F	NF	F	NF	F	NF	F
Kite strings and similar cases.....	9..	15..	7..	8..	1..	9..	17..	32
Climbing poles or towers.....	6..	8..	8..	12..	5..	25..	19..	45
Cranes, booms, well rigging.....	26..	49..	14..	46..	11..	62..	51..	157
Long pipes, beams, flag poles, and so forth.....	7..	12..	10..	23..	4..	13..	21..	48
Climbing trees and trimming.....	3..	9..	3..	5..	5..	3..	11..	17
Contacting directly or indirectly wires down.....	15..	47..	7..	33..	12..	38..	34..	118
On freight cars and outside trolleys.....	11..	8..	4..	11..	4..	20..	19..	39
Nonelectrical workers: house movers, fence builders, and so forth.....	6..	13..	3..	17..	2..	13..	11..	43
Electrical engineers, testers, electricians.....	4..	3..	4..	18..	1..	10..	9..	31
Auto accidents, felling trees, and so forth.....	3..	11..	1..	9..	6..	21..	10..	41
Primary crossed with secondary.....	0..	2..	0..	3..	0..	0..	0..	5
Miscellaneous.....	5..	10..	4..	12..	3..	11..	12..	33
Totals.....	95..	187..	65..	197..	54..	225..	214..	609

NF—Nonfatal F—Fatal

Fatal accidents reported during the year 1945 within the electric light and power industry are analyzed in the April 1947 issue of Distribution Engineering. Whereas 68 percent of the fatalities were attributed to electric shock or burns involving circuits of from 110 to 66,000 volts, only four fatalities were reported for voltages in excess of 33 kv in spite of the fact that persons work around circuits up to and including 220 kv while energized.

In general, the hazard from contact with high-voltage power circuits is appreciated by both the electrical worker and the public; unfortunately, recognition of the danger of low-voltage shocks is not equally widespread. Although it is true that the hazard from contact with electric circuits is largely dependent upon voltage, the actual danger to life depends almost entirely upon the current produced. A man accidentally coming into contact with a 60,000-volt circuit when standing on a very dry wood pole may cause a smaller current to flow than one who inadvertently grasps a defective 110-volt portable appliance when in a bathtub. Ohm's law, $I=E/R$, applies in both cases. The difference in the circuit

resistance in the two cases may be sufficient to compensate for the great difference in the voltages. It is the ratio of voltage to resistance that determines the current that will flow and the danger to life. It is difficult for the layman to grasp this difference between the hazard of voltage and the danger of electric current. Current is the proper measure of electric shock intensity; damage to living tissue is caused by current and not by voltage. For a given voltage, the current that flows in a circuit depends upon the resistance of the circuit.

With the exception of special constant current circuits, the resistance of the power circuit is usually negligible in comparison with the resistance of the body circuit; the latter includes the internal resistance of the body, skin, and contact resistance. It is believed that the resistance between major extremities, such as between the arms, leg to arm, and leg to leg, is about 500 ohms; the resistance from temple to temple about 100 ohms. The resistance of the body circuit when the skin is dry may be as high as 500,000 ohms. When the points of contact are wet with salt solutions or perspiration, this value may fall to 1,000 ohms. Furthermore, body and skin resistance decrease with both time and current. In high-voltage shocks, deep burns may be produced instantly, and the protection afforded by the skin then vanishes. In this case, the resistance of the circuit suddenly falls from its original high value to a low value of 500 ohms or less. The protection afforded by dry skin accounts for the relatively few accidents on low-voltage circuits.

It is important to note that the normal or rated current of a circuit bears no relation to the current which may flow during accidental contact or during a short circuit. The danger from electric shock resides in the current passing through the body and not in the normal current consumption of the device. It has been shown that the current passed through the body in accidental contact depends upon (1) the voltage, (2) the body, skin, and contact resistance, and (3) the resistance of the rest of the circuit (in most cases this latter item may be neglected). As moisture and firm contact with the circuit greatly affect the total resistance, the hazard is increased many times when moisture and well-grounded objects are involved. By way of contrast, consider the automobile. At the instant a driver steps on the starting switch, the 6-volt storage battery produces currents upward of 400 amperes, but there is no electric-shock hazard because of the very low voltage of the battery. By contrast, a 110-volt circuit in which no current is flowing may be extremely hazardous when hands that are wet or bleeding make contact with it. The hazard is amplified many times during contact with high-voltage circuits which are also likely to produce burns puncturing the skin resistance and thus reducing the circuit resistance to a very low value. In such instances the current developed may be sufficient to blow fuses or operate circuit breakers.

A consideration of the effects of 60-cycle alternating current will serve to indicate the various causes of death due to electric shock. The minimum current that just can be perceived is important, because it is essential that the user of an electric appliance not get the sensation of shock. Although no

electrical hazard is produced by currents slightly in excess of the threshold of perception, such shocks are startling and may cause loss of balance and subsequent injury by a fall. With gradually increasing alternating currents, the first sensations of tingling give way to contractions of the muscles. Sensations of heat and muscular contractions increase as the current is increased; then sensations of pain develop, and finally the current is such that a person cannot release his grasp of the conductor. At this point the victim is said to "freeze" to the circuit, and, if this is long continued, collapse, unconsciousness, and death result. Owing to the statistical nature of the response, and to the fact that abnormalities would be present in a large group of the general population, it is impossible to define an absolutely safe current for all persons. For many practical purposes the "let-go current" for 99 and 1/2 percent of an experimental group is considered a reasonably safe current. If connections to the body are firm or wet and the shock is of short duration, currents at the "let-go" level produce no aftereffects. Burns may be produced if abnormally large current densities are present, such as those resulting from pin-point contacts and sparks. Currents in excess of one's "let-go" limit are frightening and painful. Reasonably safe "let-go currents" for 60-cycle alternating current for men and women are 9.0 and 6.0 milliamperes, respectively. The corresponding values for direct current are 62 and 41 milliamperes. Alternating currents in excess of about 25 milliamperes are very painful, and, when the current pathway is across the chest, the muscular reactions become so severe that breathing is difficult if not impossible for the duration of the shock. Death may result from asphyxiation if the current persists for more than a few minutes. However, if the current is interrupted within a reasonable time, breathing is resumed automatically, and no serious aftereffects result.

Currents considerably in excess of those causing stoppage of breathing due to muscular contractions may produce temporary paralysis of the nerves controlling respiration, a condition termed respiratory inhibition. Respiratory paralysis may last for a considerable period after interruption of the current, and immediate and continued artificial respiration must be applied to prevent asphyxial death. Often, respiratory inhibition disappears in a few minutes, or in a few hours; therefore, the continued application of artificial respiration may save the victim. Mere cessation of natural breathing is not likely to produce serious aftereffects or permanent damage, as is evidenced by the many persons who have been resuscitated successfully.

Currents in excess of 100 milliamperes, if they take a pathway through the body in the region of the heart, may affect the heart, causing ventricular fibrillation which is nearly always fatal. For shocks short in duration (when compared with the period of the heart cycle), the probability of producing ventricular fibrillation varies with the part of the heart cycle in which the shock occurs. The greater tolerances for shocks of short duration, together with the variability in sensitivity of the heart to fibrillate, explains why some accident victims survive apparently heavy momentary shocks, such as those from capacitor discharges and high-voltage impulse generators.

The susceptibility to ventricular fibrillation increases with current up to a maximum and then decreases as the current is increased. The explanation of this phenomenon is that the muscular contractions produced by high currents cause such violent contractions of the chest and heart muscles that the heart is held still in a viselike grip, and fibrillation is prevented. If the shock is of appreciable duration, death from heart failure is inevitable; if the shock is of short duration, the muscles spontaneously relax upon interruption of the current, and the heart may resume its normal rhythm. This concurs with observed accident cases, for it has been observed that the proportion of victims of high-voltage shocks who can be resuscitated increases as the circuit voltage increases. Although there is no known cure for ventricular fibrillation, it is believed that the abdominal massage and accompanying stimulation of the heart caused by the application of artificial respiration may be beneficial in assisting the heart to regain its normal rhythm. Because it is impossible for the layman to distinguish between respiratory inhibition, ventricular fibrillation, and heart failure, he should begin artificial respiration immediately upon rescue of the victim from the circuit. It is very important that resuscitation be continued without interruption, because, if the supply of oxygen to the brain is cut off for more than a few minutes, serious permanent injury to the brain is likely to result should the victim recover. The victim should be kept warm and a physician brought to the scene as soon as possible. Artificial respiration should be continued until the victim recovers, or until rigor mortis sets in, or a physician pronounces him dead.

Possible Results of a Brief Contact With 60-Cycle Alternating Current

Body and Contact Resistance	Circuit Voltage		
	100 Volts	1,000 Volts	10,000 Volts
Low—500-1,000 ohms....	Certain slight burns	Probable death, marked burns	Possible survival severe burns
5,000 ohms.....	Painful shock, no injury	Certain death, burns probably slight	Probable death severe burns
High—50,000 ohms.....	Scarcely felt	Painful shock, no injury	Certain death burns probably slight

Relatively large currents, amperes and not milliamperes, may cause death by heating of the body, hemorrhages, and serious burns. Delayed death may be caused by burns or other serious aftereffects. Death also may result from a combination of the causes just mentioned, or from complications such as injury by a fall or a broken neck.

The table above summarizes much of the data presented in this article. It is gratifying that, in a majority of cases, victims surviving serious electrical accidents suffer no permanent disability. (Electrical Engineering, Aug. '47 - C. F. Dalziel)

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Vaccination Against Q Fever: Q fever has risen in status from a disease of localized interest in a few widely scattered areas of the world to one of considerable importance to military medicine in the Mediterranean area. Furthermore, two separate outbreaks have been recognized in the United States recently. Essentially no information is available on the mode of spread of the disease among allied personnel who were stationed in the Mediterranean Theater. The

naturally occurring disease in Australia and the United States has been limited almost entirely to workers in stock yards and packing houses. The Australian investigators state, "Meatworkers may conceivably acquire infection from cattle either directly or indirectly. In the latter case, the mode of infection may sometimes be inhalation of dust containing (infected) tick feces." It will be recalled that naturally infected ticks have been found in a number of cattle-raising areas in the United States. It is a well established fact that a high incidence of infection occurs among laboratory workers engaged in the study of Q fever.

There is a very apparent need for some means of immunological protection of exposed workers in laboratories and slaughter houses and possibly for persons in the highly endemic Mediterranean area. Vaccines capable of inducing resistance in experimental animals against infection with Rickettsia burneti have been employed in other laboratories. These were prepared from infected spleens of guinea pigs, livers and spleens of infected mice, yolk sacs, and from infected tick tissues and feces. None of these vaccines has been used for the immunization of man.

In the present study, Q fever vaccines were tested for their capacity to induce resistance in guinea pigs to infection and to elicit the production of specific complement-fixing antibodies in guinea pigs and human beings. The Henzerling (Italian) and Dyer (American) strains of R. burneti were employed. The vaccines were prepared from yolk sacs of infected eggs and formalinized by methods similar to those used in the commercial manufacture of epidemic typhus vaccine.

It was found that the vaccines were capable of eliciting demonstrable resistance to infection in guinea pigs. This resistance was most apparent when vaccinated animals were challenged with lethal amounts of R. burneti. The immunity was not absolute, however, since vaccinated animals injected with material containing many infectious doses of rickettsiae almost always developed a short, nonfatal febrile illness. Only occasionally was there encountered in immunized guinea pigs the complete resistance which has been described by others who have prepared Q fever vaccines. These results may have depended upon the use of more potent challenge material, since the mortality rates in the controls were higher than those of Bengtson. Animals immunized with vaccine prepared from either the Henzerling or Dyer strain were equally resistant to infection with the homologous and heterologous strains. This is in agreement with the work of others.

A single injection of Q fever vaccine elicited about the same response in guinea pigs as did 3 injections. The rickettsial material is relatively stable since heating at 60 C for 30 minutes did not appreciably affect its immunogenic activity. Washed suspensions of R. burneti which contained relatively small amounts of egg proteins compared with the regular vaccines, were good immunizing agents.

Guinea pigs injected with vaccine prepared from either strain developed, within 2 weeks, complement-fixing antibodies which reacted with Henzerling

antigen. However, these animals did not have detectable complement-fixing antibodies which reacted with Dyer antigen until about 4 weeks after vaccination. Both types of antibody increased rapidly following their first appearance and occurred in approximately equal amounts 8 weeks following vaccination.

None of 108 guinea pigs vaccinated in the present work failed to develop complement-fixing antibodies of Q fever. Therefore, antibodies were present in appreciable amounts in the sera of all animals which resisted infection, as well as in those few which died following challenge. The available data apparently indicate that animals which possessed circulating antibodies capable of reacting with both Henzerling and Dyer antigen were no more resistant than animals having complement-fixing substance which reacted only with Henzerling antigen.

All but two of 39 persons injected with Q fever vaccine developed specific complement-fixing antibodies. Human beings, like guinea pigs, first produce substances that react with Henzerling antigen. Unlike them, however, few persons subsequently develop Dyer antibodies. In this respect, vaccinated persons show the same type of serological response as recovered patients, since sera of the latter only occasionally react with Dyer antigen. No opportunity has arisen to obtain information on the resistance of these vaccinated individuals to infection with Q fever. (Am. J. Hyg., Jan. '48 - J. E. Smadel et al.)

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Q Fever Studies in Southern California: A previous report of observations made during the spring of 1947 on the occurrence of 17 cases of Q fever in Los Angeles County and subsequent studies of 100 additional cases indicated that Q fever is endemic in Southern California. Proximity to dairies by reason of occupation or residence was a common factor in the histories of more than 50 percent of the cases. Except for dairy workers, it was noted that the infected persons rarely used milk from nearby dairies. It was also found in fairly extensive serological surveys that from 10 to 20 percent of the dairy cows in the Los Angeles area possessed serum antibodies for Q fever.

These studies, when completed, will be reported later. The purpose of this paper is to report the recovery of Rickettsia burneti, the causative agent of Q fever, from the raw milk of four widely separated dairies in Los Angeles County.

Epidemiological data pointed to these dairies as being involved in recent human cases (dairy workers or nearby residents). Raw milk from suspected dairies was tested as being the possible source of infection. The cows were prepared for milking in the usual manner by washing the udder with water, but the udders of some were further washed with 70-percent alcohol before specimens were taken. Specimens from individual cows were hand milked into sterile wide-mouth vials which were sealed immediately after sampling all four quarters

of the udder. In some instances the specimens represented strippings taken after milking machines had been used.

Rickettsial organisms, identified by all available criteria as R. burneti, were recovered by each of three laboratories from the milk of the four dairies. A total of 50 milk specimens was injected into guinea pigs, 40 of them giving evidence of infection with R. burneti.

Injections into guinea pigs with whole blood and blood clots from more than 150 cattle, most of them lactating cows (some serologically positive) from dairies where there were human cases of Q fever, produced negative results. However, most of these specimens were shipped unfrozen to the National Institute of Health and preserved at icebox temperatures for as long as a month before they were used for inoculation.

A pool of urine and a pool of feces taken from 5 cows from one dairy were inoculated into guinea pigs. Q fever rickettsiae were not recovered from these excretions despite the fact that organisms were recovered from milk taken from the same cows at the same time. Other specimens of urine and feces have been tested with negative results; however, more extensive studies with these excretions are planned.

Four blood specimens and one spleen specimen from calves ill with fever and diarrhea of undetermined origin were tested. Inoculation experiments and serological tests of recovered calves indicated that these animals were not infected with Q fever organisms.

Pooled specimens of flies, mosquitoes, and several species of free living mites, collected from alfalfa feed were injected into guinea pigs on a limited scale. Completely negative results were obtained. Spinose ear ticks also were injected, but tests are not yet complete.

The relative ease with which R. burneti was recovered from milk of dairies in Los Angeles City and County suggests a high degree of availability of this pathogenic agent to the human and animal populations of the area, since nearly all of this milk is transported about the county before processing and much of it is sold raw. The occurrence of Q-fever infection in the human population and a demonstrable widely disseminated source of R. burneti in the same area suggest a causal relationship. Whether or not milk represents an effective source of infection to man, however, cannot be determined by the data presented in this report.

The evidence presented by outbreaks in packing houses, stockyards, and laboratories did not indicate that the drinking of infected milk was a cause of those outbreaks. A pulmonary route of infection was considered the most likely possibility in several of these outbreaks. Incomplete studies in California suggest that, for certain specific occupational and residential groups, the drinking of infected milk is an improbable mode of infection. However, the evidence did

not rule out infected milk as a potential source of infection to man by some mode yet to be determined.

The failure to recover R. burneti from whole blood, blood clots, urine, and feces of a limited number of cows shedding R. burneti in their milk and the absence of a demonstrable illness in the infected animals suggests that a local infection of the udder may occur in the absence of concurrent severe systemic infection in the cow. The presence of R. burneti was not associated, however, with observable pathology in the udder or with diminution in either quantity or quality of milk. (Pub. Health Repts., 13 Feb '48 - R.J. Huebner et al.)

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The Lymphoid Tissue and Antibody Formation: Several early investigators in immunology considered the possibility that the spleen was the main site of antibody formation, but conclusive evidence for this was not found. Hektoen, using the well-known fact that x-ray produces a destructive effect on all lymphoid tissue, demonstrated that antibody formation is inhibited to a marked degree in x-rayed animals. He concluded from this observation that the spleen, lymphoid tissue, and bone marrow were the principal sites of immune bodies. Murphy and Sturm confirmed Hektoen's findings but considered that they had eliminated the bone marrow as of first importance in the process. This conclusion was based on the fact that depletion of the lymphoid tissue was effected by repeated, small doses of x-rays of low penetration which produced no detectable damage in the bone marrow or other tissues. Yet, animals treated in this manner were found deficient in the production of antibodies. In addition, these investigators demonstrated that rabbits, after stimulation of the lymphoid system by dry heat treatment, had an enhanced ability to produce immune substances. Hussey found that if the lymphoid system of guinea pigs is kept in a depleted state by x-rays following a sensitizing injection of horse serum, the animals show no anaphylactic manifestations on receiving a second injection of serum. In such animals the antigen continues free in the circulation as long as exposure to x-rays is continued. McMaster and Hudack have clearly demonstrated that antibodies may be formed in lymph nodes, but their experiments did not eliminate the possibility that cells other than lymphocytes participated in the reaction. Ehrich and Harris have extended these observations, and on the basis that the tissue response accompanying the formation of antibodies in the nodes is predominately a lymphoid one, they consider these cells as the important factor in the development of immune substances. Harris, Grimm, Martens, and Ehrich, in a study of the antibodies in the lymph, found that when the lymphocytes are separated from the lymph plasma that the titer of the cell extract is substantially and consistently higher than that of the lymph plasma. This is taken to indicate that the lymphocytes either produce antibodies or take them up from lymph plasma. Because no evidence for the latter was found, they believe that the lymphoid cells are the responsible agents. Dougherty, Chase, and White noted that the antibody content of the blood is increased following the administration of adrenal cortical extract, a fact which

they attributed to the destructive action of the hormones on the lymphocyte with the release of the antibodies supposedly formed in the cytoplasm of these cells. Philips, Hopkins, and Freeman found no such increase when the lymphoid tissue was destroyed by nitrogen mustard, and that such treatment may result in an actual depression of the production of antibodies.

In the present investigation an estimate has been made of the antibody formation in animals with extreme hypertrophy of the lymphoid system which follows the removal of the adrenal glands. The release of the antibodies from the lymphocyte under these conditions would be due to the normal shedding of the cytoplasm as the cell matures. In the absence of the adrenals there can be no question of the cortical hormones playing any part in the reaction.

The circulating lymphocytes show a definite but not marked rise following removal of the adrenals in rabbits. This increase is in the average range of 4,000 cells per cubic mm., which is considerably less than that observed for mice and rats. However, if adrenalectomized rabbits are given an antigen, the lymphocyte count shows a marked increase up to an average of over 14,000 cells, whereas normal rabbits under the same condition have only a slight increase.

The results from the experiments carried out are essentially a confirmation of an earlier observation by the authors that animals with hypertrophied lymphoid systems have an enhanced ability to produce antibodies. Adrenalectomized rabbits with hypertrophy of the lymphoid tissues produce antibodies in amounts far in excess of that produced by intact animals. This is evident not only by definite reactions in higher dilution but by the more massive precipitate in low dilutions. The prevention of the lymphoid hypertrophy in the adrenalectomized animals by administration of adrenal cortical hormones does not reduce the amount of antibodies in the serums. This is probably due to the disruptive action of the hormone on the lymphocyte with a more rapid release of the immune globulin than would normally take place. If the immune substances are present in the cytoplasm of the lymphocyte, and there is evidence for this belief, the release into the circulation is probably due to shedding, which is known to occur as the lymphocyte matures. One of the tests previously carried out by the authors confirms the observation of Dougherty, Chase, and White that the release of antibodies may be definitely augmented by adrenal cortical hormones. The present experiments demonstrate that the hormone is not essential for this release and therefore, strictly speaking, antibody production is at least not entirely under the control of the adrenals. (Proc. Soc. Exper. Biol. and Med., Nov. '47 - J. B. Murphy and E. Sturm)

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Absorption From the Pulmonary Alveoli: The ways in which molecules of different sizes, and finally visible particles, including bacteria, are absorbed from the lungs, present recurring problems.

The routes of absorption from normal lung alveoli require passage through the epithelial lining of the alveoli and then the crossing of endothelium-lined blood capillaries into the blood or, if the second available route is to be followed, penetration of a lymphatic capillary through a delicate endothelial wall into the lymph stream. There is no doubt that bronchiolar absorption occurs, but both from the surface area available and the anatomical barriers imposed, it is of little importance compared with the absorption from the alveoli.

Experiments lasting 4 hours were carried out upon dogs anesthetized with nembutal. The right lymphatic duct and thoracic duct were cannulated and collection of lung lymph and blood specimens was accomplished after intratracheal instillation of dog plasma, purified bovine serum albumin, crystallized egg albumin, and hemoglobin. It was apparent that in 4 hours' time small protein molecules, notably serum albumin and egg albumin, found their way into the lung lymphatics. However, by using artificial respiration with a positive pressure delivery of air for inspiration, advantage was taken of the measure found most effective in promoting absorption from the alveoli into lymphatics and the flow of lung lymph.

Experiments in which pyrex glass spheres averaging 4 micra in diameter were instilled failed to disclose entrance of these distinctive foreign particles into the lymph stream, though the fact that lung phagocytes were often found containing the particles or covered with them, indicated that eventually these particles would be found in lung lymphatics and in lymph nodes.

The conclusion to be drawn is that unchanged transudates and exudates resulting from lung injury are removed from the lung alveoli in minute amounts. To clear the lungs of plasma proteins requires breakdown of molecules by enzymatic action until products are formed which are small enough to diffuse readily into the blood. The prolific supply of lymphatics in the lungs is apparently for the purpose of slowly moving wholly insoluble substances into the lymph stream, with the usual result of imprisoning them in lymph nodes prior to a possible entrance into the blood and general distribution throughout the body.

The protection against absorption from the lung alveoli is in the main due to intact alveolar epithelium through which molecules of the dimensions of the proteins commonly entering the alveoli, as a result of trauma or disease, pass very slowly and are found in small traces in lung lymph and even to a less degree in blood. When proteins are injected directly into the lung tissue and so diffuse widely in the alveolar walls, delivery of these substances by the right lymphatic duct is very prompt.

The results reported in this paper and accumulated over a number of years, accord with and extend those obtained by Cameron and Courtice and by Courtice and Phipps. (J. Exper. Med., 1 Jul '47 - C. K. Drinker and E. Hardenbergh)

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Circular Letter 48-15

9 February 1948

To: All Ships and Stations

Subj: Patient's Jackets and Clinical Records (Items 31, 41, and 43, BuMed Field Records Schedule); Instructions Concerning.

Refs: (a) Par. 12B11.5(c), ManMedDept.
(b) Par. 514.1, ManMedDept.

This letter from the Chief of BuMed (See N.D. Bulletin of 15 February 1948 for full copy) gives instructions for the disposition and handling of subject records (1) for the years prior to 1940 and (2) for the years 1940 and thereafter. Reference (b) is to be revised in the near future, to be in conformity with the instructions in this letter.

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Circular Letter 48-16

9 February 1948

To: All Stations, Continental

Subj: Receipt of Medical and Dental Supplies and Equipment Direct from Contractor; Procedures for.

Ref: (a) BuMed Circular Letter 47-107

This letter from the Chief of BuMed cancels and supersedes reference (a), states that because of revised stock control and accounting procedures established by the Bureau, certain changes are necessary relative to the distribution of the vendor's shipping document, method of invoicing, and acknowledgment of receipt of medical and dental stores received direct from the contractor, and gives instructions pertaining thereto. Also, certain instructions and information are included concerning requisitioning and accounting in the case of nonstandard items not available through regular local purchase methods.

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Circular Letter 48-17

12 February 1948

To: All Stations

Subj: Medical Training Films and Other Medical Audio and Visual Aids, Availability and Report on.

Ref: (a) BuMed Circular ltr. 47-168 dated 10 Dec 1947 and published in ND Bulletin 15 Dec 1947.

This letter from the Chief of BuMed states that the Bureau of Medicine and Surgery desires to correlate procedures relative to the procurement, use, and distribution of all medical training aids (motion pictures, slide films, lantern slides, still photography, charts, etc.) within the naval establishment and directs that all addressees furnish the Bureau with certain information in an initial report prior to 1 April 1948 and thereafter on 1 July and 31 December of each year.

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Circular Letter 48-18

12 February 1948

To: All Stations and Fleet, Force, and Area Commands.

Subj: Quarterly Report of Rodent Control Operations

Encl: 1. (HW) Outline for use by Rodent Control Officers in preparing quarterly report.

This letter from the Chief of BuMed directs that Rodent Control Officers submit a quarterly report of rodent control operations including information of general and specific interest in connection with rodent damage and destruction, rodent distribution, and any other pertinent information.

Note: See Navy Department Bulletin of 15 February for full copy of letter and copy of enclosure.

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Circular Letter 48-19

16 February 1948

To: MedOfsCom, Naval Hospitals (Continental)

Subj: Naval Hospital Instruction of Class "A" Hospital Corps School Graduates.

Encl: 1. (HW) Copy of Instruction Schedule

1. A temporary acceleration of courses of instruction has been authorized at Hospital Corps schools to promote earlier availability of Hospital Corps personnel for duty at naval hospitals during the present personnel shortage.

2. In order to insure that all Hospital Corps school graduates are adequately trained in the elementary duties of the Hospital Corps, it is directed that they be assigned to a course of instruction in practical nursing at naval hospitals as outlined in enclosure 1. Graduates are to be assigned to this course during

the first two months following the original date of reporting from Hospital Corps schools.

--BuMed. C. A. Swanson

Note: Copy of enclosure not reprinted in News Letter.

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Circular Letter 48-20

17 February 1948

To: All Ships and Stations

Subj: Mineral Oil in Dental Sterilizers

1. The substitution of mineral oil as the sterilizing liquid in dental sterilizers which are manufactured for use with water creates a potential fire hazard and shall be discontinued.
2. It is not the intent to restrict or discourage the use of hot oil as a sterilizing agent, provided it is used in a sterilizer manufactured for that purpose. Hot oil sterilization of dental instruments, especially dental handpieces, has proved satisfactory at a number of naval activities, including the U. S. Naval Dental School, National Naval Medical Center, Bethesda, Maryland.
3. Hot oil sterilizers are nonstandard items. Activities requiring them should procure them in accordance with BuMed Circular Letter No. 47-33 dated 17 March 1947.

--BuMed. H. L. Pugh

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Circular Letter 48-21

17 February 1948

To: All Ships and Stations

Subj: BuMed Circular Letters Relating to Nurse Corps Officers, Cancellation of.

1. The following BuMed Circular Letters which have served their purpose or have been superseded by later directives are hereby canceled:

<u>BuMed C/L No.</u>	<u>Date</u>	<u>N.D. Bul. Item No.</u>
46-36	6 Feb 1946	46-365
46-51	11 Mar 1946	46-574
46-67	15 Apr 1946	46-893

--BuMed. H. L. Pugh

Circular Letter 48-22

17 February 1948

To: Commandants, Eleventh, Twelfth, and Thirteenth Naval Districts
Attn: District Dental Officers

Subj: District Dental Officer Inspection Reports, Forwarding of Via Western
Sea Frontier

Ref: (a) BuMed Circular Letter No. 47-84.

This letter from the Acting Chief of BuMed cancels reference (a) which states that for the purpose of coordinating Medical Department Logistics Planning, the District Dental Officer Inspection Reports shall be forwarded via Commander, Western Sea Frontier for endorsement.

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